

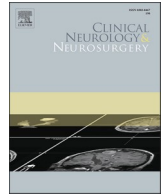


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## Case Report

# Guillain-Barre Syndrome and COVID-19: A case report

T. Bueso<sup>a,\*</sup>, V. Montalvan<sup>a</sup>, J. Lee<sup>a</sup>, J. Gomez<sup>a</sup>, S. Ball<sup>b</sup>, A. Shoustari<sup>c</sup>, P. Julayanont<sup>a</sup>, C. Jumper<sup>c</sup>

<sup>a</sup> Department of Neurology, Texas Tech University Health Sciences Center School of Medicine, Lubbock, TX, USA

<sup>b</sup> Department of Internal Medicine, Texas Tech University Health Sciences Center School of Medicine, Lubbock, TX, USA

<sup>c</sup> Department of Internal Medicine, Pulmonology and Critical Care, Texas Tech University Health Sciences Center School of Medicine, Lubbock, TX, USA

## ARTICLE INFO

### Keywords:

SARS-CoV-2

COVID-19

Guillain-Barré syndrome

Polyneuropathies

IVIG

## 1. Introduction

On December 31, 2019, a novel coronavirus, SARS-CoV-2, was identified in Wuhan, China. In a few months, it spread at an exponential rate and came to be known as the COVID-19 pandemic resulting in a devastating impact on global healthcare and economic systems. COVID-19 commonly manifests with fever, dyspnea, and cough. Interestingly, studies on COVID-19 have also reported neurological complications. Since the outbreak, multiple cases of COVID-19 patients presenting with Guillain-Barré Syndrome (GBS) have been reported [1]. GBS is an acute onset polyneuropathy that typically presents with symmetric ascending flaccid paralysis and paresthesia associated with autonomic disturbances [2]. The severe inflammatory response and the critically-ill nature of many COVID-19 patients present a challenge in distinguishing GBS from other neurologically mimicking conditions, including critical illness polyneuropathy and myopathy.

## 2. Case description

A 60-year-old Caucasian woman with history of migraines presented with fever, non-productive cough, myalgia, and dysgeusia for 10 days. A nasopharyngeal swab for SARS-CoV-2 RT-PCR assay was positive. She was admitted for worsening dyspnea. Upon admission, chest computed tomography revealed “ground-glass” opacities. She was started on supplemental oxygen, azithromycin, and hydroxychloroquine. Twenty-two days after the onset of viral symptoms, she developed sacro-lumbar pain,

feet numbness, and weakness in her legs for which she required a walker for ambulation. Within two days, the weakness symmetrically progressed to the involvement of both the lower and upper extremities to the point that she became bed-ridden during her hospital stay. Respiratory function worsened with increasing oxygen requirements through a Venturi mask at 8 L/minute. Her heart rate (HR) and systolic blood pressure (BP) fluctuated between 70–140 beats per minute and 100–180 mmHg, respectively throughout short periods.

Neurological examination revealed symmetrical weakness with Medical Research Council (MRC) grade 2 out of 5 with areflexia in both proximal and distal muscle groups of the lower extremities and MRC grade 3 out of 5 with hyporeflexia (1+) on both proximal and distal muscle groups at the upper extremities. The strength of neck flexion and extension was MRC grade 3 out of 5. All sensory modalities were normal. The straight leg raise test was positive bilaterally. The Erasmus GBS Respiratory Insufficiency Score at initial evaluation was three, which correlated to a 17 % risk of developing respiratory failure in the first week of admission. During her hospital stay, she was noted to have a sodium level of 122 meq/L secondary to a syndrome of inappropriate antidiuretic hormone secretion (SIADH), which improved following fluid restriction and administration of salt tablets and furosemide. She also developed dysautonomia with fluctuation in HR and mean arterial pressure, as well as transient fecal incontinence and urinary retention. Spirometry revealed a negative inspiratory force (NIF) of –35 cm water and a forced vital capacity of 1.7 L. Cerebrospinal fluid (CSF) analysis revealed cytoalbuminologic dissociation (CAD) with 197 mg/dL of

\* Corresponding author.

E-mail address: [tulio.bueso@ttuhsc.edu](mailto:tulio.bueso@ttuhsc.edu) (T. Bueso).

<https://doi.org/10.1016/j.clineuro.2020.106413>

Received 16 May 2020; Received in revised form 21 November 2020; Accepted 26 November 2020

Available online 5 December 2020

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proteins and 0 white blood cells. Her clinical manifestations and laboratory findings were consistent with GBS. The patient was started on intravenous immune globulin (IVIG) 0.4 g/kg/day for 5 days in addition to enoxaparin 30 mg twice a day. After a week of therapy, the patient showed improvement in both her respiratory and neurological function. Two months after admission the patient was followed up as an outpatient; she was found ambulating with assistance, an MRC grade 4 was assessed throughout her muscle groups, and persistent neuropathic pain in her lower extremities.

### 3. Discussion

GBS is a disorder in which the immune system attacks gangliosides on the peripheral nervous system. The immune response damages either myelin (acute inflammatory demyelinating polyradiculoneuropathy, AIDP) or the axon (acute motor axonal neuropathy, AMAN, and acute motor and sensory axonal neuropathy, AMSAN) [2]. Multiple cases have been reported associating COVID-19 and GBS, most patients initially presented with paresthesia and progressive quadriparesis. Acute polyneuropathy triggered by the coronavirus infection has been reported mostly in critically ill patients. The involvement of the peripheral nervous system has been observed in patients with a confirmed diagnosis of COVID-19 providing evidence for the proposed coronavirus neurotropic invasion pathway [1]. It is still unclear whether SARS-CoV-2 can directly invade neurons and cause neuropathy. While SARS-CoV-2 in CSF could not be assessed in our case, the absence of white blood cell in this patient's CSF indicated that acute polyneuropathy was more likely an immune response typically seen in GBS rather than direct neuronal invasion of the virus, as in the latter case pleocytosis would be expected.

Prior cases of COVID-19 patients with GBS did not demonstrate SIADH or dysautonomia. However, the patient in our case had dysautonomia symptoms, including BP and HR variability, fecal incontinence, and urinary retention. Dysautonomia has also been linked to an inappropriate secretion of ADH and renin by blockage of normal conduction in sinoatrial stretch receptors [3]. Dysautonomia is associated with a higher disability score and a significantly higher risk of mortality in patients with GBS. Spirometric parameters such as the vital capacity need to be frequently monitored in patients with GBS due to possible development of restrictive pulmonary dysfunction secondary to diaphragmatic denervation.

A combination of ventilation/perfusion mismatch and restrictive

respiratory failure can be expected in COVID-19 patients with GBS, and ventilator-weaning criteria should be individualized in these critically ill patients [4].

### 4. Conclusion

Our case emphasizes that in addition to respiratory failure from acute respiratory distress syndrome in COVID-19 patients, neurologic complications, such as GBS, should be considered as one of the differential diagnosis in patients with polyneuropathy and difficulty weaning off the ventilator. The ultimate goal is for clinicians to be able to make an early diagnosis of GBS in complicated or critically ill COVID-19 patients for them to have the best chance of successful recovery.

### Authors contribution

TB: Conceptualization, Data curation, Writing- Original draft preparation. VM: Conceptualization, Writing- Original draft preparation. JL: Writing- Original draft preparation. JG: Writing- Original draft preparation. SB: Writing, Editing. AS: Writing, Editing. PJ: Writing- Reviewing and Editing. CJ: Writing, Editing.

### Acknowledgments

To the nurses, respiratory technicians, and University Medical Center Medical Intensive Care Unit staff, which perform a selfless, compassionate, and unrelenting service to the community of Lubbock, Texas during this current COVID-19 pandemic. Dr. Kenneth Nugent for his editorial assistance.

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